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FILE 'CAPLUS' ENTERED AT 22:57:40 ON 09 NOV 2008
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FILE 'MEDLINE' ENTERED AT 22:57:40 ON 09 NOV 2008

FILE 'USPATFULL' ENTERED AT 22:57:40 ON 09 NOV 2008
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FILE 'BIOSIS' ENTERED AT 22:57:40 ON 09 NOV 2008
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=> s (NK1 (5A) (receptor(3A)antagonist))
L5        2399 (NK1 (5A) (RECEPTOR(3A) ANTAGONIST))

=> s L5 (P) (COPD or (chronic (W)obstructive (W) pulmonary(W) (disease or
disorder)))
L6           5 L5 (P) (COPD OR (CHRONIC (W) OBSTRUCTIVE (W) PULMONARY(W) (DISEA
               SE OR DISORDER)))

=> dup rem L6
PROCESSING COMPLETED FOR L6
L7            4 DUP REM L6 (1 DUPLICATE REMOVED)

=> s L7 NOT Pd>20020708
L8           1 L7 NOT PD>20020708

=> d L8 TI AB IBIB

L8   ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   SCH 206272: a potent, orally active tachykinin NK1, NK2, and NK3 receptor
     antagonist
AB   Expts. were performed to characterize the pharmacol. of SCH 206272
     [(R,R)-1'[5-[(3,5-dichlorobenzoyl)methylamino]-3-(3,4-dichlorophenyl)-4(Z)-
     (methoxyimino)pentyl]-N-methyl-2-oxo-[1,4'bipiperidine]-3-acetamide] as a
     potent and selective antagonist of tachykinin (NK) NK1, NK2, and NK3
     receptors.  SCH 206272 inhibited binding at human tachykinin NK1, NK2, and
     NK3 receptors (Ki=1.3, 0.4, and 0.3 nM, resp.) and antagonized [Ca2+]i
     mobilization in Chinese hamster ovary (CHO) cells expressing the cloned
     human tachykinin NK1, NK2, or NK3 receptors.  SCH 206272 inhibited
     relaxation of the human pulmonary artery (pKb=7.7.+-.0.3) induced by the
     tachykinin NK1 receptor agonist, [Met-O-Me] substance P and contraction of
     the human bronchus (pKb=8.2.+-.0.3) induced by the tachykinin NK2 receptor
     agonist, neurokinin A.  In isolated guinea pig tissues, SCH 206272
     inhibited substance P-induced enhancement of elec. field stimulated
     contractions of the vas deferens, (pKb=7.6.+-.0.2), NKA-induced
     contraction of the bronchus (pKb=7.7.+-.0.2), and senktide-induced
     contraction of the ileum.  In vivo, oral SCH 206272 (0.1-10 mg/kg, p.o.)
     inhibited substance P-induced airway microvascular leakage and neurokinin
     A-induced bronchospasm in the guinea pig.  In a canine in vivo model, SCH
     206272 (0.1-3 mg/kg, p.o.) inhibited NK1 and NK2 activities induced by
     exogenous substance P and neurokinin A.  Furthermore, in guinea pig models
     involving endogenously released tachykinins, SCH 206272 inhibited
     hyperventilation-induced bronchospasm, capsaicin-induced cough, and airway
     microvascular leakage induced by nebulized hypertonic saline.  These data
     demonstrate that SCH 206272 is a potent, orally active tachykinin
     NK1, NK2, and NK3 receptor antagonist.  This
     compd. may have beneficial effects in diseases thought to be mediated by
     tachykinins, such as cough, asthma, and chronic
     obstructive pulmonary disease.
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ACCESSION NUMBER: 2002:668660 CAPLUS
DOCUMENT NUMBER: 138:265440
TITLE: SCH 206272: a potent, orally active tachykinin NK1, NK2, and NK3 receptor antagonist
AUTHOR(S): Anthes, John C.; Chapman, Richard W.; Richard, Christian; Eckel, Stephen; Corboz, Michel; Hey, John A.; Fernandez, Xiomara; Greenfeder, Scott; McLeod, Robbie; Sehring, Susan; Rizzo, Charles; Crawley, Yvette; Shih, Neng-Yang; Piwinski, John; Reichard, Greg; Ting, Pauline; Carruthers, Nick; Cuss, Francis M.; Billah, Motasim; Kreutner, William; Egan, Robert W.
CORPORATE SOURCE: Department of Allergy, Schering-Plough Research Institute, Kenilworth, NJ, 07033, USA
SOURCE: European Journal of Pharmacology (2002), 450(2), 191-202
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier Science B.V.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 44 THERE ARE 44 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> FIL STNGUIDE

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	27.38	243.37

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-0.80	-5.60

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FILE CONTAINS CURRENT INFORMATION.
LAST RELOADED: Nov 7, 2008 (20081107/UP).

=> file caplus, medline, uspatfull, biosis

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	0.48	243.85

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	0.00	-5.60

FILE 'CAPLUS' ENTERED AT 23:04:30 ON 09 NOV 2008
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FILE 'MEDLINE' ENTERED AT 23:04:30 ON 09 NOV 2008

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FILE 'BIOSIS' ENTERED AT 23:04:30 ON 09 NOV 2008
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```
=> s (NK1 (5A) (receptor(3A)antagonist))
L9        2399 (NK1 (5A) (RECEPTOR(3A) ANTAGONIST))

=> s L9 (P) (anticholinergic or bronchodilat? or (M3 (3A) muscarinic(2A)antagonist))
L10         17 L9 (P) (ANTICHOLINERGIC OR BRONCHODILAT? OR (M3 (3A) MUSCARINIC
               (2A) ANTAGONIST))

=> s L10 and scopine
L11          0 L10 AND SCOPINE

=> dup rem L10
PROCESSING COMPLETED FOR L10
L12          17 DUP REM L10 (0 DUPLICATES REMOVED)

=> s L12 NOT Pd>20020708
L13          5 L12 NOT PD>20020708

=> d L13 1-5 TI AB IBIB

L13  ANSWER 1 OF 5  CAPLUS  COPYRIGHT 2008 ACS on STN
TI   Pharmacology of MEN 11467: a potent new selective and orally-effective
     peptidomimetic tachykinin NK1 receptor antagonist
AB   We have investigated the pharmacol. properties of MEN 11467, a novel
     partially retro-inverse peptidomimetic antagonist of tachykinin NK1
     receptors.  MEN 11467 potently inhibits the binding of [3H] substance P
     (SP) to tachykinin NK1 receptors in the IM9 lymphoblastoid cell line (pKi
     = 9.4.+-.0.1).  MEN 11467 is highly specific for the human tachykinin NK1
     receptors, since it has negligible effects (pKi <6) on the binding of
     specific ligands to tachykinin NK2 or NK3 receptors and to a panel of 30
     receptors ion channels unrelated to tachykinin receptors.  The antagonism
     exerted by MEN 11467 at tachykinin NK1 receptors is insurmountable in
     satn. binding expts., both KD and Bmax of SP were significantly reduced by
     MEN 11467 (0.3-10 nM).  In the guinea-pig isolated ileum, MEN 11467
     (0.03-1 nM) produced a nonparallel rightward shift of the concn.-response
     curve to SP methylester with a concomitant redn. of the Emax to the
     agonist (pKB = 10.7.+-.0.1).  Moreover the antagonist activity of MEN
     11467 was hardly reversible despite prolonged washout.  In vivo, MEN 11467
     produced a long lasting (> 2-3 h) dose-dependent antagonism of
     bronchoconstriction induced by the selective tachykinin NK1 receptor
     agonist, [Sar9, Met(O2)11]SP in anesthetized guinea-pigs (ID50s' =
     29.+-.5, 31.+-.12 and 670.+-.270 .mu.g/kg, after i.v., intranasal and
     intraduodenal administration, resp.), without affecting
     bronchoconstriction induced by methacholine.  After oral administration
     MEN 11467 produced a dose-dependent inhibition of plasma protein
     extravasation induced in guinea-pig bronchi by [Sar9, Met(O2)11] (ID50 =
     6.7.+-.2 mg/kg) or by antigen challenge in sensitized animals (ID50 = 1.3
     mg/kg).  After i.v. administration MEN 11467 weakly inhibited the GR
     73632-induced foot tapping behavior in gerbil (ED50 = 2.96.+-.2 mg/kg),
     indicating a poor ability to block central tachykinin NK1 receptors.
     These results demonstrate that MEN 11467 is a potent, highly selective and
     orally effective insurmountable pseudopeptide antagonist of peripheral
     tachykinin NK1 receptors with a long duration of action.
ACCESSION NUMBER:        2002:484021  CAPLUS
DOCUMENT NUMBER:         137:379900
TITLE:                   Pharmacology of MEN 11467: a potent new selective and
                         orally-effective peptidomimetic tachykinin NK1
                         receptor antagonist
AUTHOR(S):               Cirillo, R.; Astolfi, M.; Conte, B.; Lopez, G.;
                         Parlani, M.; Sacco, G.; Terracciano, R.; Fincham, C.
                         I.; Sisto, A.; Evangelista, S.; Maggi, C. A.; Manzini,
```

S.
CORPORATE SOURCE: Department of Pharmacology, Menarini Ricerche SpA, Pomezia-Roma, I-00040, Italy
SOURCE: Neuropeptides (Edinburgh, United Kingdom) (2001), 35(3&4), 137-147
CODEN: NRPPDD; ISSN: 0143-4179
PUBLISHER: Elsevier Science Ltd.
DOCUMENT TYPE: Journal
LANGUAGE: English
REFERENCE COUNT: 32 THERE ARE 32 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L13 ANSWER 2 OF 5 CAPLUS COPYRIGHT 2008 ACS on STN
TI In vitro and in vivo pharmacology of S 16474, a novel dual tachykinin NK1 and NK2 receptor antagonist
AB Since tachykinins released from lung sensory nerve endings are thought to play a role in inflammatory diseases of airways via NK1 and NK2 receptors, dual tachykinin NK1 and NK2 receptor antagonists may have a great therapeutic potential. In vitro, the cyclopeptide S 16474 (cyclo-[Abo-Asp(D-Trp(SucONa)-Phe-N-(Me)Bzl)]) bound to both human tachykinin NK1 and NK2 receptors expressed in two lines of transfected Chinese hamster ovary cells (IC50 values 85 nM and 129 nM, resp.), while showing a poor affinity for the rat tachykinin NK1 receptor. S 16474 inhibited the contractions induced by substance P in isolated rabbit vena cava (pA2 7.0) and by neurokinin A in rabbit pulmonary artery (pA2 5.6). In vivo in anesthetized guinea-pigs, S 16474 was found to dose dependently inhibit the bronchoconstrictions induced by i.v. administered substance P, neurokinin A and capsaicin. Plasma extravasation evoked in bronchi by endogenously released tachykinins under vagus nerve stimulation was abolished by S 16474 (10 .mu.mol/kg i.v.). These results demonstrate clearly that S 16474 is a tachykinin receptor antagonist exhibiting, in vitro and in vivo, a dual inhibitory effect on NK1 and NK2 receptors.
ACCESSION NUMBER: 1996:7149 CAPLUS
DOCUMENT NUMBER: 124:136392
ORIGINAL REFERENCE NO.: 124:25139a,25142a
TITLE: In vitro and in vivo pharmacology of S 16474, a novel dual tachykinin NK1 and NK2 receptor antagonist
AUTHOR(S): Robineau, Pascale; Lonchampt, Michel; Kucharczyk, Nathalie; Krause, James E.; Regoli, Domenico; Fauchere, Jean-Luc; Prost, Jean-Francois; Canet, Emmanuel
CORPORATE SOURCE: Division de Pneumologie, Institut de Recherches Servier, 11 Rue des Moulineaux, Suresnes, F-92150, Fr.
SOURCE: European Journal of Pharmacology (1995), 294(2/3), 677-84
CODEN: EJPHAZ; ISSN: 0014-2999
PUBLISHER: Elsevier
DOCUMENT TYPE: Journal
LANGUAGE: English

L13 ANSWER 3 OF 5 CAPLUS COPYRIGHT 2008 ACS on STN
TI In vitro and in vivo biological activities of SR140333, a novel potent non-peptide tachykinin NK1 receptor antagonist
AB SR140333 (I) is a new non-peptide antagonist of tachykinin NK1 receptors. SR140333 potently, selectively and competitively inhibited substance P binding to NK1 receptors from various animal species, including humans. In vitro, it was a potent antagonist in functional assays for NK1 receptors such as [Sar9,Met(O2)11]substance P-induced endothelium-dependent relaxation of rabbit pulmonary artery and contraction of guinea-pig ileum. Up to 1 .mu.M, it had no effect in bioassays for NK2 ([.beta.Ala8]neurokinin A-induced contraction of

endothelium-deprived rabbit pulmonary artery) and NK3 ([MePhe7]neurokinin B-induced contraction of rat portal vein) receptors. The antagonism exerted by SR140333 toward NK1 receptors was apparently non-competitive, with pD2' values (antagonism potency evaluated by the neg. logarithm of the molar concn. of antagonist that produces a 50% redn. of the maximal response to the agonist) between 9.65 and 10.16 in the different assays. SR140333 also blocked in vitro [Sar9,Met(O2)11]substance P-induced release of acetylcholine from rat striatum. In vivo, SR140333 exerted highly potent antagonism toward [Sar9,Met(O2)11]substance P-induced hypotension in dogs (ED50 = 3 .mu.g/kg i.v.), bronchoconstriction in guinea-pig (ED50 = 42 .mu.g/kg i.v.) and plasma extravasation in rats (ED50 = 7 .mu.g/kg i.v.). Finally, it also blocked the activation of rat thalamic neurons after nociceptive stimulation (ED50 = 0.2 .mu.g/kg i.v.).

ACCESSION NUMBER: 1994:124815 CAPLUS
DOCUMENT NUMBER: 120:124815
ORIGINAL REFERENCE NO.: 120:21801a,21804a
TITLE: In vitro and in vivo biological activities of SR140333, a novel potent non-peptide tachykinin NK1 receptor antagonist
AUTHOR(S): Emonds-Alt, Xavier; Doutremepuich, Jean Daniel; Heaulme, Michel; Neliat, Gervais; Santucci, Vincent; Steinberg, Regis; Vilain, Pol; Bichon, Daniel; Ducoux, Jean Philippe; et al.
CORPORATE SOURCE: Sanofi Rech., Montpellier, F-34184, Fr.
SOURCE: European Journal of Pharmacology (1993), 250(3), 403-13
CODEN: EJPHAZ; ISSN: 0014-2999
DOCUMENT TYPE: Journal
LANGUAGE: English

L13 ANSWER 4 OF 5 CAPLUS COPYRIGHT 2008 ACS on STN
TI Effects of an NK1 receptor antagonist, FK888, on constriction and plasma extravasation induced in guinea pig airway by neurokinins and capsaicin
AB The effects of FK888, an NK1 receptor antagonist, on airway constriction and airway plasma extravasation induced by neurokinins and capsaicin were investigated in guinea pigs. FK888 inhibited substance P (10-8M)- and neurokinin A (10-9M)-induced contraction of isolated guinea pig trachea, with IC50 values of 3.2 .times. 10-8 and 4.2 .times. 10-6M, resp. FK888 given i.v. inhibited substance P (13.5 .mu.g kg-1)-induced airway constriction with an ED50 value of 0.40 mg kg-1 but did not inhibit neurokinin A (1.1 .mu.g kg-1)- and capsaicin (3.1 .mu.g kg-1)-induced airway constriction at a dose of 1 mg kg-1. On the other hand, FK888 given i.v. inhibited airway plasma extravasation induced by substance P (1.3 .mu.g kg-1), neurokinin A (11 .mu.g kg-1) and capsaicin (100 .mu.g kg-1) with equal potency and ED50 values of 0.011, 0.0063 and 0.019 mg kg-1, resp. When FK888 was given locally (into the airway directly) inhibitory activities were more potent than following i.v. administration. In this case FK888 inhibited substance P-, neurokinin A- and capsaicin-induced airway constriction with ED50 values of 3.2, 190 and 550 .mu.g kg-1, resp., suggesting that an about 100 times higher dose is required to inhibit neurokinin A- and capsaicin-induced airway constriction than substance P-induced constriction. FK888 given orally was also effective in substance P-, neurokinin A- and capsaicin-induced airway plasma extravasation with ED50 values of 4.2, 5.9 and 9.5 mg kg-1. These results demonstrate that FK888 is an effective in vivo NK1 receptor antagonist and the different inhibitory activity of FK888 on airway responses suggests that substance P-, neurokinin A- and capsaicin-induced airway plasma extravasation is solely mediated via NK1 receptors whereas in airway constriction only substance P-induced reaction is mediated via NK1 receptors.

ACCESSION NUMBER: 1993:462785 CAPLUS

DOCUMENT NUMBER: 119:62785
ORIGINAL REFERENCE NO.: 119:11089a,11092a
TITLE: Effects of an NK1 receptor antagonist, FK888, on constriction and plasma extravasation induced in guinea pig airway by neurokinins and capsaicin
AUTHOR(S): Murai, Masako; Maeda, Yasue; Hagiwara, Daijiro; Miyake, Hiroshi; Ikari, Norihiro; Matsuo, Masaaki; Fujii, Takashi
CORPORATE SOURCE: Dep. Pharmacol., Fujisawa Pharm. Co., Ltd., Osaka, 532, Japan
SOURCE: European Journal of Pharmacology (1993), 236(1), 7-13
CODEN: EJPHAZ; ISSN: 0014-2999
DOCUMENT TYPE: Journal
LANGUAGE: English

L13 ANSWER 5 OF 5 MEDLINE on STN
TI Effect of an NK1 receptor antagonist (CP-99,994) on hypertonic saline-induced bronchoconstriction and cough in male asthmatic subjects.
AB To investigate the role of NK1 receptors in the pathogenesis of bronchoconstriction and cough in asthma, we performed a randomized, double-blind, crossover study on the effects of a selective non-peptide tachykinin NK1 receptor antagonist (CP-99,994) on baseline measures of lung function and on hypertonic saline-induced bronchoconstriction and cough in 14 male subjects with mild asthma. CP-99,994 (250 micrograms/2 hours) and placebo were administered intravenously in 2-h infusions during consecutive visits 5 to 7 d apart. Specific airway resistance (SRaw) was measured and spirometry was performed at baseline and at 35 and 60 min. Next, hypertonic saline challenge was performed by delivering 10 breaths of saline of increasing concentration (0.9 to 7% in 1% increments at 5-min intervals) via an ultrasonic nebulizer until SRaw increased from baseline by 200% or 20 units, whichever was greater. Throughout the challenge cough was counted from a taped record made from two microphones placed close to the subject's larynx. We found that CP-99,994 did not significantly affect SRaw or spirometric measures of lung function during the first hour of infusion. Although CP-99,994 infusion markedly attenuated the bronchoconstrictor response to the saline challenge in two subjects, it did not significantly decrease the area under curves obtained for SRaw and cough during saline challenge for the group as a whole (p = 0.9 for SRaw;p = 0.8 for cough). We conclude that administration of 250 micrograms/kg of CP-99,994 over 2 h does not significantly inhibit hypertonic saline-induced bronchoconstriction or cough in subjects with mild asthma and does not have acute bronchodilator activity in these subjects.
ACCESSION NUMBER: 1995392865 MEDLINE
DOCUMENT NUMBER: PubMed ID: 7663799
TITLE: Effect of an NK1 receptor antagonist (CP-99,994) on hypertonic saline-induced bronchoconstriction and cough in male asthmatic subjects.
AUTHOR: Fahy J V; Wong H H; Geppetti P; Reis J M; Harris S C; Maclean D B; Nadel J A; Boushey H A
CORPORATE SOURCE: Department of Medicine, University of California, San Francisco 94143, USA.
SOURCE: American journal of respiratory and critical care medicine, (1995 Sep) Vol. 152, No. 3, pp. 879-84.
Journal code: 9421642. ISSN: 1073-449X.
PUB. COUNTRY: United States
DOCUMENT TYPE: (CLINICAL TRIAL)
Journal; Article; (JOURNAL ARTICLE)
(RANDOMIZED CONTROLLED TRIAL)
(RESEARCH SUPPORT, NON-U.S. GOV'T)

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LANGUAGE:           English
FILE SEGMENT:       Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH:        199510
ENTRY DATE:         Entered STN: 20 Oct 1995
                    Last Updated on STN: 20 Oct 1995
                    Entered Medline: 6 Oct 1995


=> d his

     (FILE 'HOME' ENTERED AT 22:49:43 ON 09 NOV 2008)

     FILE 'REGISTRY' ENTERED AT 22:51:32 ON 09 NOV 2008
L1              STRUCTURE UPLOADED
L2            0 S L1 SSS SAM
L3            8 S L1 SSS FULL

     FILE 'CAPLUS' ENTERED AT 22:52:48 ON 09 NOV 2008
L4            6 S L3

     FILE 'CAPLUS, MEDLINE, USPATFULL, BIOSIS' ENTERED AT 22:57:40 ON 09 NOV
     2008
L5         2399 S (NK1 (5A) (RECEPTOR(3A)ANTAGONIST))
L6            5 S L5 (P) (COPD OR (CHRONIC (W)OBSTRUCTIVE (W) PULMONARY(W) (DIS
L7            4 DUP REM L6 (1 DUPLICATE REMOVED)
L8            1 S L7 NOT PD>20020708

     FILE 'STNGUIDE' ENTERED AT 22:59:37 ON 09 NOV 2008

     FILE 'CAPLUS, MEDLINE, USPATFULL, BIOSIS' ENTERED AT 23:04:30 ON 09 NOV
     2008
L9         2399 S (NK1 (5A) (RECEPTOR(3A)ANTAGONIST))
L10          17 S L9 (P) (ANTICHOLINERGIC OR BRONCHODILAT? OR (M3 (3A) MUSCARI
L11           0 S L10 AND SCOPINE
L12          17 DUP REM L10 (0 DUPLICATES REMOVED)
L13           5 S L12 NOT PD>20020708

=> d que L6
L5         2399 SEA (NK1 (5A) (RECEPTOR(3A) ANTAGONIST))
L6            5 SEA L5 (P) (COPD OR (CHRONIC (W) OBSTRUCTIVE (W) PULMONARY(W)
                (DISEASE OR DISORDER)))


=> d que L10
L9         2399 SEA (NK1 (5A) (RECEPTOR(3A) ANTAGONIST))
L10          17 SEA L9 (P) (ANTICHOLINERGIC OR BRONCHODILAT? OR (M3 (3A)
                MUSCARINIC(2A) ANTAGONIST))
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